Commentary

Is non-alcoholic steatohepatitis caused by alcohol?

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Can nash be caused by alcohol

As opposed to ALD, the definition of NAFLD and NASH generally allows the daily use of <20 g alcohol. This definition – particularly in alcohol-sensitive patients – ignores the contribution of even low amounts of alcohol to the pathogenesis of NAFLD/ NASH and at the same time in somewhat relaxed attitude towards the recommended <20 g daily alcohol restriction. In fact, the recommendation of alcohol restriction in patients of NAFLD/ NASH should be an
absolute alcohol abstinence. This view is strongly supported by a recent publication demonstrating that the moderate alcohol use of 10–19 g/ day doubles the risk of advanced liver disease as compared to a daily alcohol use of 0–9 g [6]. Therefore, only alcohol abstinence would scientifically justify the term NAFLD/ NASH.

Recently, intestinal microbiota have been implicated as a potential source of hepatotoxic oxidative injury and changes in the microbiome have been shown to play a role in lipotoxicity and pathogenesis of NAFLD. Studies suggest that the specific composition of intestinal microbiota may play a role in both the inflammatory and fibrosis responses in patients with NAFLD [7]. Among 57 patients with biopsy–proven NAFLD, those with Bacteroides genus counts in the second and third tertile had a 2–fold increase in NASH compared with those with lower Bacteroides counts who were found to also have an abundance of Prevotella bacteria. With respect to fibrosis stage, those with Ruminococcus counts in the third tertile were found to have a 2–fold increase in stage 2 or greater fibrosis compared with those with lower levels of Ruminococcus. Specific gut microbiome signatures were linked to the severity of NAFLD and degree of fibrosis in additional studies [8,9]. A specific microbial metabolite, 3-(4-hydroxyphenyl) lactate, correlated significantly with hepatic fibrosis and specific bacterial species (Firmicutes, Bacteroidetes, and Proteobacteria) [8]. One proposed mechanism pertains to the endogenous production of alcohol and acetaldehyde. Colonic bacteria and yeast possess an enormous metabolic capacity for generating both ethanol and acetaldehyde, and can oxidize ethanol to high levels of acetaldehyde, even at low ethanol concentrations. Acetaldehyde is easily absorbed into the portal system.

The most compelling evidence for a contribution of alcohol to NAFLD/ NASH has recently been provided by Yuan, et al. [10]. In an individual with a severe NASH the authors discovered an ultrahigh blood alcohol concentration (BAC) of about 400 mg/ L after the consumption of a high–carbohydrate, alcohol–free diet. Further analyses revealed the endogenous alcohol production by a Klebsiella pneumoniae (Kpn) strain through fermentation of glucose/ sucrose (glucose–fructose dimer). This clinical situation was termed, Autobrewing syndrome’ (ABB) caused by a high–alcohol (HiAlc) producing Kpn strain (Figure 1). The patient eventually recovered after dietary changes and antibiotic treatment.

The further investigation of a cohort of patients with NAFLD identified a strong association between disease severity and the existence HiAlc Kpn isolates. In a HiAlc–Kpn–induced mouse model of fatty liver disease (FLD) the authors demonstrated that HiAlc–Kpn might be an important cause for NAFLD via the induction of endogenous alcohol production. Finally, transplant of HiAlc–Kpn into mice was shown to cause NAFLD. Taken together, these data provide compelling evidence that HiAlc–Kpn exist in humans and can be associated with the development of NAFLD/ NASH (Figure 2).

**Summary and recommendations**

In NALFD/ NASH alcohol abstinence is to be recommended because the intake of even low daily amounts of alcohol (< 20 g) can carry the risk of an advanced liver disease [6] while 10–19 g alcohol per day doubles the risk of advanced liver disease as compared to 0–9 g daily alcohol intake.

Recent data further suggest that the intestinal microbiome can play a major role in the pathogenesis of NAFLD/ NASH.
through an excess endogenous alcohol production [10]. While the study demonstrates that HiAlc-Kpn is a primary causative agent of NAFLD it represents only one type of etiology among other mechanisms, presumably including further roles of the microbiota that need to be investigated (Geographical Abstract, 10).

Dedication

Dedicated to the 50th birthday of Prof. Dr. med. Robert Thimme. Medical Director, Clinic of Internal Medicine II, University Hospital Freiburg.

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References


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